WHAT IS CLAIMED IS:

1. A method for monitoring the effectiveness of an administered agent that interacts with the A_3 adenosine receptor (A3AR) in ~~a~~ treatment of a disease state in an individual, the method comprising

(i) at a defined time point following administration of the agent to the individual, selected such so as to permit the agent to reach and affect cells in the individual that are associated with the disease state, withdrawing a sample of said or tissue containing said cells from the;

(ii) detecting the level of at least one physiological parameter of at least one biological marker in said cells, the marker being an A3AR,or an element associated with the A3AR signal transudation pathway downstream to A3AR; and

(iii) comparing the level of said at least one parameter to a control level, being the level thereof in such cells or tissue from the same individual before administration of said agent, or being a standard reference for said marker

which is indicative of a n un treated disease state;

wherein a difference in level of the physiological parameter from control being indicative of the effectiveness of said treatment against these state.

2. The method according to claim 1 wherein the agent that interacts with the A3AR is an A3AR agonist.

3. A method according to claim 1 wherein the A3AR signal transduction pathway is the Wnt pathway.

4. A method according to claim 3 wherein the element is at least one element selected from: PKA, PKB/Akt, GSK-3β, β-catenin, cyclin D1, c-myc.

5. A method according to claim 1 wherein the A3AR signal transduction pathway is the NF-κB pathway.

6. A method according to claim 5 wherein the element is at least one element selected from: NF-κB, PI3K, IKK, c-myc, cyclin D1.

7. A method according to claim 1 wherein the physiological parameter is selected from: the level of mRNA or protein expression, the level of phosphorylation and the cellular localization.

8. The method of claim 1, wherein said disease state is a proliferative-related disease.

9. The method of claim 8, wherein said disease is cancer.

10. The method of claim 9, wherein said cancer is melanoma, colon carcinoma or prostate cancer.

11. The method of claim 8, wherein said disease is an inflammatory disease.

12. The method according to claim 8, wherein effective treatment against the disease is indicated by a change in a physiological parameter of a biological marker selected from:

(a) a decrease of the protein level or the mRNA level coding therefore of at least one of A3AR, PKB/Akt, PKA, β-catenin, c-myc, cyclin D1 and NF-κB, TNF-α; or increase in the protein level or mRNA coding therefore of GSK-3β

(b) at least one change in phosphorylation level selected from: decrease in phosphorylation level of GSK-3β ,increase in the phosphorylation level of PKB/Akt, PKA or β-catenin.

(c) at least one change in cellular localization selected from: decrease in the localization of A3AR receptor in the cellular membrane as compared to control, decrease in the

localization of β-catenin or NF-κB in the nucleus as compared to cytosol.

13. A method according to claim 1, wherein said disease state is a disease or condition wherein a beneficial therapeutic effect is evident by increase proliferation.

14. The method of claim 13, wherein said disease state is a decrease in white blood cell count, especially neutrophils as a result of chemo- or radio-therapy.

15. The method of claim 13, wherein effective treatment against the disease is indicated by a change in a physiological parameter of a biological marker selected from:

(a) increase of the protein level or of the level of mRNA coding therefore of at least one of A3AR, PKB/Akt, PKA, β-catenin, c-myc, cyclin D1 and NF-κB, or decrease in the protein or mRNA level of GSK-3β;

(b) at least one change in phosphorylation level selected from: increase in phosphorylation level of GSK-3β, decrease in the phosphorylation level of PKB/Akt, PKA or in the phosphorylation level of β-catenin.

(c) at least one change in cellular localization selected from: increase in the localization of A3AR receptor in the cellular membrane as

compared to control, increase in the localization of β-catenin in the nucleus as compared to cytosol.

16. A method according to claim 1, wherein the level of the at least one physiological parameter of the at lease one biological marker is determined at a time point after the administration of the agent, wherein the differences between the level of the parameter in the treated subject and the untreated control are expected to be the most prominent.

17. A method according to claim 2 wherein the A3AR agonist is 1-deoxy-1-[6[[(3-iodophenyl)methyl]amino]-9H-purine-9-yl]-N-methyl-β-D-ribofura-nuronaminde (IB-MECA).

18. A method for determining whether a drug candidate is an A3AR agonist useful in treating a disease state manifested in diseased cells, the method comprising:

(i) obtaining a sample of the diseased cells or tissue containing said cells;

(ii) contacting said sample with the drug candidate;

(iii) detecting the level of at least one physiological parameter of at least one biological marker in said cells, the marker being an A3AR, or an element associated with the A3AR signal transudation pathway which is downstream to the A3AR; and

(iv) comparing the level of said at least one parameter to the level thereof in untreated sample not contacted with said drug candidate;

wherein a difference in level of the physiological parameter between the treated and untreated sample being indicative that the drug candidate is an agonist of A3AR.

19. A method according to claim 18 wherein the A3AR signal transduction pathway is the Wnt pathway.

20. A method according to claim 19, wherein the element is at least one element selected from: PKA, PKB/Akt, GSK-3β, β-catenin, cyclin D1, c-myc.

21. A method according to claim 18 wherein the A3AR signal transduction pathway is the NF-κB pathway.

22. A method according to claim 21 wherein the element is at least one element selected from: NF-κB, PI3K, IKK, TNF-α, c-myc, cyclin D1.

23. A method according to claim 18 wherein the physiological parameter is selected from: the level of mRNA or protein expression, the level of phosphorylation and the cellular localization.

24. The method of claim 18 wherein said disease state is a proliferative-related disease.

25. A method for determining whether a drug candidate is an A3AR agonist useful in treating a disease state manifested in diseased cells, the method comprising:

(i) administering said drug candidate to a subject having said disease state;

(ii) at one or more defined time points following the administration, withdrawing a sample of the diseased cells or tissue containing said cells from the subject;

(iii) detecting the level of at least one physiological parameter of at least one biological marker in said cells, the marker being an A3AR, or an element associated with the A3AR signal transudation pathway which is downstream to the A3AR; and

(iv) comparing the level of said at least one parameter to the level in diseased cells withdraw from a subject not administered with said drug candidate;

wherein a difference in level of the physiological parameter between the treated and untreated sample being indicative that the drug candidate is an agonist of A3AR.

26. A method according to claim 25 wherein the A3AR signal transduction pathway is the Wnt pathway.

27. A method according to claim 26, wherein the element is at least one element selected from: PKA, PKB/Akt, GSK-3β, β-catenin, cyclin D1, c-myc.

28. A method according to claim 25 wherein the A3AR signal transduction pathway is the NF-κB pathway.

29. A method according to claim 22 wherein the element is at least one element selected from: NF-κB, PI3K, IKK, TNF-α, c-myc, cyclin D1.

30. A method according to claim 25 wherein the physiological parameter is selected from: the level of mRNA or protein expression, the level of phosphorylation and the cellular localization.

31. The method of claim 25 wherein said disease state is a proliferative-related disease.